great where no quinine has been administered as where quinine has been administered. On the other hand quinine has been found in heavy concentration in the spinal fluid of the new-born infant, and it must be seriously considered whether fetal idiosyncracy to quinine may not be responsible for congenital deafness in certain cases, and perhaps in many more cases than have ever been suspected. This matter is receiving experimental investigation, for, in spite of the great value of quinine in obstetrics, the tragedy of congenital deafness is so serious that obstetricians might well find other methods of sensitizing the uterus to contraction than by the use of quinine.

Through the American Otological Society efforts are being made in the United States and in Canada to correlate the research now being conducted so that the results in one laboratory are being checked in another and so that there is as little duplication of endeavour as possible. The experimental investigation of deafness is one of the most useful and promising fields of medical investigation of the day.

CONCLUSION

Finally, from the all-important view point of the education of the child, it is necessary to stress the necessity of recognizing defective hearing at the earliest possible age. Every infant is born dumb, but irremediable deafness at birth is rare. Later on, due to the sense of hearing, the child acquires articulate language. Infants who have some degree of deafness are comparatively common, but not infrequently the mother can so make use of the hearing a child possesses in the training of the child that it grows up with fairly useful hearing; but every infant born totally deaf will remain dumb. In the pre-school age hearing plays a decisive rôle in the learning of speech. The child does not learn to speak properly unless it has good hearing, and such ability to speak as it has may be lost or not maintained if it develops a hearing defect. The hard-of-hearing school-child suffers a very severe handicap, which is the more severe because frequently neither the parent nor the school teacher nor the physician has appreciated that the apparent stupidity of the child has its cause in deafness. Slight but significant hearing impairment is often the cause of retardation, a speech defect, an inferiority complex, or even unusual behaviour. Many children who have not been recognized as deaf or hard-of-hearing fail at school, and have to turn to other channels because they cannot keep up with their school mates. Educational authorities have discovered that there are several times as many repeaters among the hard-of-hearing children as among normal children, and that therefore the deaf child is an expensive burden. Along with this discovery they are becoming more and more awake to the fact that the handicap of deafness segregrates the deaf child from the normal child and makes him less fit to develop into a helpful citizen in his adult years.

POLIOMYELITIS: VITAMIN B DEFICIENCY A POSSIBLE FACTOR IN SUSCEPTIBILITY*

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DRAPER has defined poliomyelitis as "an acute infectious disease in the course of which paralysis is but an accidental and incidental occurrence". Aycock estimates that in the worst epidemics 1 person in 500 contracts poliomyelitis; but, if "abortive" and non-paralytic cases are included, it may be one in ten. In other words, there may be fifty mild or subclinical cases to one frank paralytic case. In a recent epidemic in Greenland (1932) Hrolv[1] reported 83 pronounced cases in a total population of 2,263, resulting in 20 deaths. He states that during the epidemic "nearly all the inhabitants complained of headache and indisposition for two or three days, particularly the children, many of whom had headache with moderate constipation and mild fever". It was his belief that these persons were more or less infected without presenting definite symptoms of acute invasion or signs of paralysis. A previous epidemic in Greenland (1914) resulted in

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37 deaths in a population of 700. At that time the outbreak began with an "influenza-like" disease which was so general that the reindeer hunt of the season was a complete failure. Paralysis was observed in some of the hunters after their return home.

In the more recent epidemic in Toronto, during the past summer, in a population of approximately 650,000, there were 747 cases reported, 183 of which exhibited paralytic symptoms, resulting in 30 deaths. Two-thirds of the cases reported were under 10 years of age, and three-fifths were in males. During this epidemic there were doubtless hundreds, and possibly thousands, of subclinical or potential "polio" cases, of the "summer flu" type, that responded to the usual home treatment without medical attention.

With the accruing evidence from repeated epidemics it becomes increasingly obvious that a much smaller proportion of the reported cases actually suffer paralytic involvement, as compared with the earlier history of the disease, when the paralytic cases only were recognized. It would therefore appear that the hypothetical virus of this disease, which has in the past been regarded as specifically neurotropic, is little more selective in its action on the central nervous system than the infectious agents of other diseases, notably diphtheria, malaria and influenza.

Myers[2] reports 1,316 cases of diphtheria with 275 developing paralysis, and 80 deaths, most of the latter being due to cardiac paralysis. Of the paralytic cases, as to age, 104 were under 5 years, 138 were from 5 to 10 years and 33 were above 10 years. There was a marked preponderance of paralysis among the males. The paralysis usually comes on during convalescence. Occasionally, however, it occurs in the early stage of the disease, even on the 2nd or 3rd day. The close parallelism in these figures as to age, sex and paralysis incidence, when compared with the poliomyelitis figures in the recent Toronto epidemic, is most striking.

Zingher,[3] in a report on over one thousand cases of poliomyelitis under 5 years of age, found over 80 per cent to be Schick-positive. Baginsky says: "I have been struck with the very extraordinary sensitivity of children suffering with such diseases of the central nervous system as poliomyelitis, spastic cerebrospinal paralysis, hemiplegia, etc. One is scarcely able to keep these children in the hospital wards free from infection with diphtheria." The situation in this respect at the present time may be somewhat altered owing to the general adoption of toxoid immunization.

In a comparative study of poliomyelitis and diphtheria Jungeblut[4] reports poliocidal substances occasionally demonstrable in the serum of monkeys actively immunized against diphtheria toxin. He and his associates also found that contact *in vitro* with adrenalin, cortin and vitamin C is equally destructive for both the toxin of diphtheria and the virus of poliomyelitis. In a discussion of the predisposition of certain constitutional types to severe forms of diphtheria, Seckel reports six cases with five deaths, two of which patients were previously attacked by poliomyelitis; while Wernstedt reports three cases of poliomyelitis, one fatal, which developed in children within 11 to 19 days after the onset of diphtheria. In reviewing this subject Jungeblut concludes that the data presented "are strongly suggestive of the operation of some common basic mechanism which governs susceptibility and resistance to both poliomyelitis and diphtheria". Obviously there must be some constitutional factor which determines the age and sex incidence and susceptibility to paralysis.

Draper has emphasized the frequency with which certain constitutional types are found in poliomyelitis wards. He notes specially the prevalence of wide interpupillary spaces, high-arched palates, cleft upper incisor teeth, etc., with the inference of a possible endocrine imbalance as an endogenous factor in susceptibility. With reference to this factor he further states: "The highly specialized type of child described above is a causal factor of equal importance with the virus in the occurrence of infantile paralysis; but, so far as development of paralysis is concerned, the constitution of the child is of greater significance than the virus."

On the assumption of possible exogenous factors in susceptibility, based on nutritional imbalance, the recent work of Jungeblut and Toomey is of interest. The former, in experimental poliomyelitis in monkeys, has obtained a fair degree of protective effect from vitmain C when administered within a rather definite range of dosage. The latter has demonstrated increased susceptibility when vitamin D is withheld in conjunction with a diet otherwise rich

in vitamins. But, strange to say, little or no work of a conclusive nature has been done in this respect with the anti-neuritic vitamin B. With the established anti-paralytic effect of vitamin B in beriberi it would seem that this pioneer in the vitamin field should offer the most hope of protective value in poliomyelitis.

Recent reports of successful treatment of many common paralytic conditions by vitamin B give support to this hypothesis. Favourable results have attended the administration of vitamin B concentrates in the treatment of alcoholic polyneuritis,[5] post-diphtheritic paralysis,[6] neuritic paralyses associated with pernicious vomiting of pregnancy (based on the belief that a fetus increases the demands on the mother for vitamin B, while persistent emesis decreases the maternal intake), and the paralyses of diabetes and pernicious anæmia,[7] referable to impoverished diet. Swineford[8] reports paralysis in a prolonged typhoid case, in which administration of vitamin B brought about prompt relief of the neuromuscular symptoms. Vorhaus and his associates[9] report the favourable treatment of 100 cases of neuritis with 10 mg. of crystalline vitamin B daily. Three of their cases which were associated with pregnancy were completely relieved of their neuritic symptoms. Gerstenberger[10] reports remarkably favourable results in the vitamin B treatment of herpes, a neuropathy referable to a neurotropic virus and classified pathologically as a posterior poliomyelitis. Hoobler[11] reports a favourable response to vitamin B therapy in a number of cases of toxæmia in infants associated with muscular rigidity. Gruenfelder and his associates[12] interpreted a form of infantile toxæmia occurring in Jerusalem in the hot weather as a form of vitamin B deficiency, and obtained surprisingly good results with vitamin B concentrates intravenously.

In a comparative study of poliomyelitis and beriberi many features common to both diseases provide a striking similarity. In poliomyelitis mostly infants and juveniles are attacked, with a noticeable predilection for males, while beriberi has its greatest incidence in infancy and in young adult males. Poliomyelitis has its peak of incidence in the hot weather—July, August and September in northern temperate climates—and the corresponding summer months in southern temperate climates; while beriberi prevails in tropical climates where the weather is warm almost constantly. Both diseases are predominantly characterized by flaccid paralysis of the leg muscles, with ascending and bulbar varieties involving the cranial nerves, and with very similar neuropathology. Gastro-intestinal disturbances and muscle tenderness are characteristic of both diseases, œdema is pathologically common to both, and physical over-exertion is a well recognized predisposing factor in both.

A striking illustration of the possible basic relationship of poliomyelitis and beriberi is provided in the epidemiological history of Nauru, a small volcanic island in the southern Pacific ocean. Previous to the Great War this little island of eight square miles area was a German colonial possession, but has been under mandate to Australia since 1919. The population in 1910 consisted of 1,250 native Nauruans, about 500 natives of the neighbouring Carolines, about 500 Chinese, and 80 Europeans. The last three groups were all employees of a phosphate mining company, the sole industry of the island.

In 1905-6 the German colonial physician reported a number of cases of "infectious polyneuritis" on the island, followed in 1910 by what is regarded as the most intensive epidemic of poliomyelitis on record. Within 14 days some 700 cases developed, resulting in 38 deaths. The greatest incidence of the disease was among the native Nauruans, with 470 cases and 37 deaths. There were 220 cases with one death among the Carolinese phosphate workers, these all being treated in the company's hospital. The Chinese were completely exempt, and among the Europeans there were only three mild cases. The onset of all cases was with fever and chills followed by digestive disturbances, constipation and sensation of pressure or fullness of the stomach (a symptom common in beriberi, due to gastric atony). Vomiting, diarrhœa and headache were also common. Pain in the back of the neck, lumbar region and legs was often noted, all the muscles being very sensitive to pressure. Paræsthesia and paralysis developed in most cases, usually disappearing during convalescence, although at the end of three months there were some 50 cases of pronounced residual paralysis. Œdema of the legs below the knees was noted in a number of cases (suggestive of wet beriberi). In most of the fatal cases respiratory paralysis developed. Diplopia was noted in two cases and œsophageal paralysis in one case. The pulse rate, compared to the fever, was noticeably accelerated (a feature common to beriberi).

The age-incidence of the epidemic was somewhat unusual for poliomyelitis, most cases being within the limit of 12 to 40 years. The old people were completely exempt, as were most of the young children, in spite of constant contacts. Three nursing infants were afflicted along with their mothers (such cases are rarely noted in poliomyelitis, but not uncommon in beriberi).

A striking feature as to incidence was the fact that among the Carolinese workers of the phosphate company nearly all the severe paralyses occurred in a group that came from the island of Singelap, although they constituted but 15 per cent of the Carolinese workers. A noticeable constitutional characteristic of these, as in the Nauruans, was a "spongy fattiness" (suggestive of general cellular œdema, indicative of a beriberi background).

The transmission of the disease to the island could not be established. Vessels from Australia, which had been 11 days in transit, had no cases on board; and steamers from Hong Kong, nearly three weeks en route, had no cases, and neither was there any similar disease

at ports of call. Natives arriving by these steamers were and remained healthy through the entire epidemic.

Owing to the unusual epidemiological features of this outbreak the local colonial physician, Dr. A. Müller, was hesitant in identifying the disease as poliomyelitis. Aside from the neuromuscular phenomena he regarded the epidemic as typical of influenza, especially the rapid spread and almost simultaneous incidence of the cases. However, some years later many of the victims of this epidemic were very carefully examined and found to have residual paralyses and deformities characteristic of poliomyelitis, and at the present time the identity of the disease is generally regarded as established.

In October, 1920, the post-war pandemic of pneumonic influenza swept the island, with an incidence of 100 per cent and a mortality rate of 30 per cent. In the trail of this holocaust ill health and debility were everywhere manifest, a condition which was apparently aggravated by an obvious dietary deficiency. To make matters worse, leprosy broke out and spread so rapidly in spite of the usual precautionary measures that by 1925 fully one-third of the population exhibited clinical signs of the disease.

In 1926 Dr. G. W. Bray, the new medical officer of health under the Australian régime, who had noticed that for a number of years previously the native infant mortality under one year of age had been almost 50 per cent of the total births, made an investigation of the dietetic habits of the natives in an effort to remedy the situation. Previous to this time the sale of polished rice had been forbidden, and the use of fermented "toddy", made from the sap of the cocoanut tree, had also been interdicted, but no restriction was placed on the consumption of sugar, white flour and canned meats, all of which were void of vitamin B. Dr. Bray had noticed that most of the infant mortality had occurred in breast-fed babies during the third month from birth, and that this condition did not apply to infants separated from their leprous mothers and fed artificially by relatives. Most of these infant deaths had been recorded as due to "stomach trouble", "infantile diarrhœa", "convulsions", "marasmus" and "broncho-pneumonia", but Dr. Bray concluded from a careful analysis of the situation that the basic ailment was infantile beriberi, similar to the type so often seen in Japan and the Philippines. Accordingly, samples of the mothers' milk were tested chemically and biologically. The protein and fat were found to be low and the sugar high, due perhaps to the habitual use of sugar-water by the native women as a beverage. Samples of the milk fed to chickens produced typical polyneuritis in about three weeks, which was readily cured by feeding yeast. Based on the obvious assumption of a vitamin B deficiency, a radical change in the native dietary was instituted. The sale of white flour and polished rice was forbidden in the native stores, whole-meal flour and brown rice being substituted, and the use of whole wheat bread was encouraged. Sweet potatoes were introduced, and the value of eggs and condensed milk was stressed. The sale of sugar was restricted to 1 lb. per adult per week. The use of the yeast-laden cocoanut-sap beverage was again permitted, and the sick babies were treated with an emulsion of the yeast in cod-liver oil. As a result of these measures the infant death rate rapidly fell from 50 to 7 per cent, at which level it has consistently remained. Not only has there been no further reported occurrence of "poliomyelitis" or infantile beriberi, but even leprosy and tuberculosis, which were rife on the island, have been almost completely eradicated. This is in harmony with the observation of Takaki, that whenever beriberi was reduced in the Japanese navy the health of the men improved generally and other diseases decreased.

The most obviously logical conclusion from the above narrative is that vitamin B deficiency furnished the basic background for most of the neuropathological conditions of this unfortunate island. It, perhaps, may have accounted for the unprecedented high rate of incidence of the 1910 epidemic of "poliomyelitis", and may have contributed to the unusually high mortality of the 1920 epidemic of influenza—a disease also noted for its effect on the nervous system. It was probably also the chief etiological factor in the "infectious polyneuritis" of 1905-6.

On the assumption of B hypovitaminosis as common to poliomyelitis and beriberi the deficiency may conceivably be precipitated in the former as a sequel to the acute infectious invasion, in which the febrile state, by greatly increasing the metabolic demand for the vitamin, brings about a rapid depletion of its storage in the tissues; whereas in the latter the vitamin deficiency is generally of slow and insidious onset as a result of long continued dietetic error and prolonged action of predisposing factors. Under these conditions it is quite conceivable that the degree of destructive action on the central nervous system might be materially different, the protective responses of the organism having more time to act in beriberi than in acute poliomyelitis. There are not a few instances, however, in which a febrile infectious invasion has precipitated beriberi. Bentley[13] reports 52 cases of beriberi in which 28 appear to have been the sequel of recurring malaria or dysentery and allied conditions. Three cases of beriberi following relapsing fever are reported by Yacoub, associated with prolonged feeding on condensed milk, a food containing a scant amount of vitamin B. In Brazil beriberi is so frequently associated with a preliminary febrile condition, of an influenza-like nature, that clinicians in that country still regard the disease as basically infectious.

There are a number of determining factors in relation to the vitamin B requirement of the organism which have a direct bearing on the question of its possible deficiency in poliomyelitis. In the first place it has been shown by Gaynor and Dennett[14] that the storage of vitamin B in the tissues of young children is very limited, excess supplies of the vitamin being quickly exhausted from the system. Hendricks[15] and Cowgill[16] have shown that the vitamin B requirement bears a direct relation to the metabolic rate. According to DuBois, with increased physical exertion and in all febrile states there is a marked increase in the metabolic rate. Accordingly, under these conditions there would be a corresponding depletion of the tissue storage of vitamin B. In fevers associated with gastro-

intestinal disturbances, such as in beriberi and poliomyelitis, the depletion of the anti-neuritic vitamin is not only thereby hastened but the ingestion and assimilation of new supplies is cut off. The metabolic rate has its highest peak at the period of most rapid growth and activity, in childhood, declining gradually with maturity and advancing age. The metabolic rate is also higher in the male. It is, therefore, perhaps not without significance that the age and sex ratios of the metabolic rate, and consequently the corresponding ratio of the vitamin B requirement, bear an almost parallel relationship to the age and sex incidence of paralysis in poliomyelitis, diphtheria and beriberi. The seasonal influence of gastro-intestinal disturbances in children, such as "summer flu", "stomach flu" and "summer complaint", with their depleting effect on vitamin B tissue storage, may also be a contributing factor in the seasonal incidence of poliomyelitis. Another factor related to vitamin B demand and seasonal incidence may be the marked increase in growth and physical activity of children during the school holidays in the summer months. Prof. W. T. Porter, of Harvard University, found in the observation of 3,000 school children that the seasonal period of greatest growth and weight increase was during the summer and early fall.

In view of all these factors contributing to vitamin B depletion it seems not unreasonable to conclude that an acute deficiency of this essential anti-neuritic element may so condition the nervous system that the infectious agent of poliomyelitis may more readily effect destructive action resulting in paralysis.

An interesting sidelight in this connection is the reported observation of concurrent epidemics of paralytic diseases in domestic animals, notably dog "distemper" and encephalomyelitis in horses, sheep and cattle. It is not, perhaps, merely coincidental that these animal diseases, which exhibit pathological changes in the central nervous system almost identical with poliomyelitis in man, also have relatively the same age, sex and seasonal incidence. Even the ancient Greeks and Romans recognized the seasonal incidence of disease, notably during the "dog days" (dies caniculares) in July and August, associated with the influence of Sirius the dog star. Hesiod tells us that "Sirius parches head and knees" (typical of poliomyelitis). Homer speaks similarly, calling it the evil star, the star of late summer. The pestilences which then prevailed occasioned the offering of sacrifices to placate the inimical star.

On the basis of the deficiency hypothesis advanced herein many of the hitherto unexplained features of poliomyelitis may be somewhat clarified. Aside from the age, sex and seasonal incidence, which seem definitely related to the metabolic demand for vitamin B, it would account for the spotty and relatively small incidence of paralytic symptoms, the conspicuously noticeable lack of contagious relationship between such cases, and the recognized ineffectual control by quarantine. Davison[17] says: "Isolation for three weeks of patients and contacts is required by most boards of health since 1916, but the evidence then and now does not indicate that anything is accomplished by this procedure . . . The disease rarely attacks more than one member of a family, and cases developed from contact are conspicuously rare. Of 2,070 persons definitely exposed only 14 contracted the disease . . . patients with the non-paralytic or subclinical type of the disease must be so common that nearly the whole population should be isolated."

In the last quarter century there has been a gradual advance in the age line in poliomyelitis. The cases under five years, in New York City, in 1907, were 86.8 per cent; in 1916, 79.2 per cent; in 1931, 53.3 per cent; in 1935, 32.8 per cent. In Philadelphia the cases under five years were, in 1916, 71.1 per cent; in 1932, 51.5 per cent. A similar advance has been noted in the Toronto epidemics. Improved infant hygiene, particularly the adaptation of the newer knowledge of the vitamins to the dietetics of infancy and childhood, has significantly paralleled this advance in the age line.

In recent years considerable prominence has been given in both the medical and commercial world to the importance of the vitamins, particularly the A, C and D varieties, as reflected in the increased use of fish-liver oils and citrous fruits, irradiation of food products, and exposure of the body to ultra-violet lamps and sunlight; but vitamin B seems to have been left in the background. The infant and young child of today are amply supplied with vitamins A, C and D in the form of cod-liver oil, orange and tomato juice. A scant supply of vitamin B is supplied in both human and cow's milk; but no routine measures are employed to contravene vitamin B deficiency in the diet of young children at a time when rapid growth makes increased demands for this essential food element. McCollum points out that there is very clear evidence that nutritive disorders

have a far-reaching influence in controlling the health of children, bringing about many border-line cases of malnutrition. He emphasizes the danger to health in adherence to a diet in which milled cereal products, particularly white bread, and sugar, syrup, tubers and meat of the muscle type predominate (all deficient in vitamin B). Williams says: "The discovery of the vitamins has entirely altered our conceptions of the causes and origins of disease. Until lately disease was regarded as a sin of commission by some unseen and subtle agency. The vitamins are teaching us to regard it, in some degree at any rate, as a sin of omission on the part of civilized or hypercivilized man. By our habit of riveting our attention on microbes and their toxins we had sadly neglected the side of the question which concerns itself with our own defences."

SUMMARY AND CONCLUSIONS

An attempt has been made to interpret the incidence of paralysis in poliomyelitis as referable in some degree to an acute deficiency of vitamin B. This, it is thought, may be brought about, in border-line cases of hypovitaminosis, by age, sex and seasonal factors determining an increase in the metabolic demands for the said vitamin, combined with the precipitating, vitamin-depleting influence of an acute systemic invasion by an infectious agent of known neurotropic properties. No experimental or clinical data are submitted in support of the theory advanced, but, if its basis is physiologically sound, it would appear that a fair clinical trial should be given to this natural nerve-protective agency which has proved to be so effectual in the treatment of other paralytic conditions of undoubted similarity. The use of this simple remedial agency would not be fraught with any of the dangers which have attended the use of serological and chemical agencies.

It is expected that the most hopeful application of the theory advanced should be in prophylaxis and during convalescence, rather than in the acute invasional stage of the disease, since the onset is so rapid that neuromuscular damage often occurs before the disease is recognized.

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PRURITUS ANI*

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PRURITUS ani may be defined as an irritative condition of the skin in the peri-anal region. Numerous causes have been suggested as the etiological factors and several classifications of the condition have been offered. I should like to submit the following practical classification of three groups.

Group one: in which there is no gross or microscopic change in the peri-anal skin, *i.e.*, in which itching is the only symptom. The common *direct* causes of this variety are: (1) lack of cleanliness; (2) certain types of underwear; (3) the presence of an irritable discharge from the rectum; (4) idiosyncrasies to certain foods; (5) parasites. The *indirect* causes are those referred from infection of the prostate (urethra), vagina, cervix, and, rarely, from a lower abdominal viscus, and neurosis. This group makes up about 5 per cent of all cases.

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